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Ph. Schwartz, M.D.

Modern Trend in the Treatment of Appendicitis
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CONTENTS

ORIGINAL ARTICLES

Birth Injury as a Cause of "Status Marmoratus" — Ph. Schwartz, M.D. — 45

Modern Trends in the Treatment of Appendicitis — Daniel H. Manfredi, M.D. — 67

Obituary—Harold Ruckman Mixsell, M.D. — 72

PEDIATRICS AT THE TURN OF THE CENTURY

Congenital Laryngeal Stridor Apparently due to an Enlarged Thymus Gland — A. W. Myers, M.D. — 73

ABSTRACTS

Wysham, D. N. et al.: Staphylococcal Infections in an Obstetric Unit. I.—Epidemiologic Studies of Pyoderma Neonatorum 83

Geyer, M. L. and Yankauer, A.: Detection of Hearing Loss in Preschool Children 84

Ying-K'un, F. and Ming-Hsun, T'an: The Sturge-Kalischer-Weber Syndrome. A Report of Four Cases 84

Paquet, E. and DeLage, J.: Rheumatic Manifestations in Hodgkin's Disease, Leukemia and Allied Conditions 85

Ricci, G.; Lo Sardo, S. and Casini, E.: Treatment of Tetanus with Chlorpromazine 85

BOOK REVIEWS

Management of the Handicapped Child. Edited by Harold Michael-Smith, Ph.D. 86

Comparative Aspects of Haemolytic Disease of the Newborn. By G. Fulton Roberts, M.D... 87

Pediatric Roentgenology. By M. A. Lassrich, M.D...Prof. R. Prévôt...K. H. Schäfer, M.D. 88

ITEMS

Myasthenia Gravis: Four Cases 66

The Prognostic Significance of Neonatal Convulsions 71

Duodenal Enzymes in Infants with Kwashiorkor or with Dystrophic Conditions 82

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ARCHIVES OF PEDIATRICS

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BIRTH INJURY AS A CAUSE OF "STATUS MARMORATUS"

PH. SCHWARTZ, M.D.*
Warren, Pa.

I. HISTORICAL NOTE

The literature on "status marmoratus" begins with a publication by Anton (1896) who found peculiar changes in the basal ganglia in a case with unusual disturbances of motility. The patient had a normal birth and a normal early development; at the age of nine months scarlet fever occurred; he died before reaching adolescence. At autopsy, both putamina were interspersed with white patches resembling myelinated structures. The grayness of the putamina persisted only in scattered spots. The microscopic examination showed that this change was restricted entirely to the putamina. Although Anton emphasized that birth and early development were normal, he found it necesary to write the following: "I don't intend to discuss in great detail those interesting observations of symmetrical brain lesions due to birth trauma. . . . Symmetrically located hemorrhages in the brain may originate from . . . circulatory disturbances associated with veins . . . in the brain." We assume that these remarks have more than incidental meaning. Anton may have believed that there was a connection between birth injury and the condition he defined.

Our knowledge, however, has advanced in another direction. In 1895, Oppenheim described the clinical symptoms of a 31-year-old woman who exhibited bilateral athetosis, general rigidity, and a typical speech disorder ("congenital pseudobulbar paralysis"). She had an illegitimate daughter who, born after difficult labor, developed identical symptoms, and died at 23 years of age. Anatomical examinations of the brain, performed by C. Vogt (1911), revealed alterations of the striate nuclei similar to those described

* Chief, Department of Pathology, Warren State Hospital, Warren, Pa.; Professor of General Pathology and Pathological Anatomy, University Frankfurt-Main, Germany; Former Director of the Pathologic Institute at the University of Istanbul, Turkey.

by Anton. In a second case, too, which was clinically observed by Freund, C. Vogt disclosed an "état marbré" in the caudate nucleus and putamen. All other parts of the brain apparently were intact. The birth was normal, but there was the history of an "apoplexy" in early infancy. In subsequent publications (1918, 1919, 1920) C. Vogt and O. Vogt developed the hypothesis that "status marmoratus," along with other conditions, was a "genotypical form of a malformation."

In one of his first papers on birth injury, published 33 years ago, the present writer pointed out that disturbances in the venous circulation at birth may be important in the causation of "status marmoratus." Simultaneously, two other contributions appeared. Scholz (1924) reported two cases in which "status marmoratus" seemed to originate from exogenous damage (infection and trauma). The most important finding of Scholz's was the evidence of a scarification process in the region of the striatum characterized by the "marbled" appearance. Thus, according to Scholz, "status marmoratus" may develop as a sequel of exogenous injury which initially induces softening in the nervous tissue and ends in scar formation containing both glia bundles and irregular myelinated nerve fibers. The other case to be mentioned here was reported by Pfeiffer (1925). A feebleminded boy, originally observed by Anton, was born prematurely by the use of forceps in a state of extreme debility, and died at the age of 15, in 1897, that is one year after the appearance of Anton's memorable publication. At the autopsy, Anton undoubtedly recognized the anatomic peculiarities of this case which confirmed his above quoted allusions to birth trauma. Although Anton did not publish this observation, he preserved the brain for almost thirty years. In Pfeiffer's paper the description, as well as the picture illustrating it, indicated that the lesion had been caused by birth trauma. Also, Pfeiffer related his interpretation to corresponding observations of the present author.

In 1927, the present writer pointed out that conditions resembling "status marmoratus" and caused by birth trauma also occur in the thalamus, in the cerebral cortex, and in the white matter of the cerebral hemispheres.

Oscar and Cécile Vogt, in 1928, recognized birth traumatic lesions as an important general factor in the etiology and pathogenesis of "status marmoratus." Since then, the tendency to explain

"status marmoratus" of various brain parts as the sequel of circulatory disturbances at birth has progressively increased. [Lowenberg and Malamud (1933); Malamud (1950); Courville (1950)]. Meyer and Cook (1936) pointed out that among twenty-three cases of "état marbré" which had been anatomically verified, seven had no birth history given; and nine of the remaining sixteen showed definite signs of birth lesion. We are indebted to Meyer and to Norman for the thorough and convincing investigations related to birth traumatic "status marmoratus."

II. On Injury of the Brain at Birth

1. *Factors Present at Delivery Which Tend to Damage the Head of the Newborn*

If we apply a suction cup on any part of the surface of our body, we observe the development of a swelling the size of which is dependent on the pressure difference between the atmosphere and the interior of the suction apparatus. If these pressure differences are great enough, the suction swelling may even be infiltrated by hemorrhages.

During the birth process similar conditions are present. Before the onset of labor, the body of the child is under equal pressure on all sides. Upon the rupture of the amnion, the intra-uterine pressure is released at that portion of the body not in contact with the uterine wall. Consequently, the body fluids will tend to stream into the region of the lesser pressure. In this way the birth swelling (caput succedaneum) is formed, some times with hemorrhages. We find release bleedings in breach presentation located in the gluteal area; in dorsal presentations, on the back; in head presentations in the scalp, in the skull and in the brain.

Of course, during birth, which even normally is a rather dangerous process, common mechanical influences also act as traumatic stresses. The bilateral narrowing of the skull, from side to side as well as fronto-occipital, during the passage of the head through the birth channel, may cause tears in the falx and tentorium. (*Beneke; L. Mayer; Hauch; Holland; Greenwood*).

As many publications have shown, the bones of the skull may become dislocated, fractured, overlapped, or depressed, even in spontaneous delivery. An abnormal presentation—forceps, version and manual extraction—enhances the development of this type of lesion.

We do not feel that all the problems of the birth mechanism and its influence on the newborn are completely solved. Nevertheless, we consider the results of experiments conclusive in which the use of a suction cup has produced changes in the surface and contents of the skull, quite similar to those observed in birth trauma.(1)

(1). In order to test the effect of lower pressure on the head and on the brain, we, together with Berberich, performed the following experiment: A suction cup was placed on the head of a newborn dog, on an area comparable to normal birth presentation in children. The suction produced by the apparatus, negative pressures from 100 to 250, also corresponded to human deliveries. We applied continual or alternating suction periods, in which the negative pressure slowly rose to a peak and then dropped, just as in normal labor. Then we diminished the suction power to a level which fastened the suction cup only slightly, imitating in this way the quiet periods between the labor pains. We were able to observe through the glass wall of our suction cup the development of a "caput succedaneum." We saw the deformation of the whole head, caused by the movements of the skull bones. We even observed the overlapping of the parietal bones pressed togther by the powerful suction. Therefore, it seems clear that the moulding of a newborn's head may occur without any interference from the walls of the birth channel and is determined solely by the suction effect. When, in the case of a narrow pelvis, the deformation of the head is severe, it is because the presented and released surface is relatively small and the process of delivery produces very high pressure differences. (S. Poeck's observations).

Some of our animals died in the experimental "birth" process. Others survived and were examined one to three days after "birth." The autopsies showed all typical birth traumatic alterations usually present in newborns: hemorrhages in the skull, bleedings in the periosteum, in the pia, and extra vasations in the brain substance itself. We observed thrombosis of the superior longitudinal sinus, the straight sinus and of its intra-cerebral tributaries. In two cases, typical fresh foci of necrosis were found localized in the fronto-parietal white substance and in the white matter of the occipital lobe. These experiments were repeated by Berberich and Stern, reproducing the bleedings in the inner ear which are characteristic of newborn infants. Thielemann also succeeded in provoking birth traumatic lesions of the inner ear. Metzger, with the same method, produced typical hemorrhages in the retina of newborn animals.

In an endeavour to produce circulatory disturbances similar to those which occur in the consequence of birth traumatic lesions, Schlesinger ligated the Galenic vein. He succeeded in provoking typical subependymal bleedings in the supply region of the terminal vein.

The differences in pressure between the interior of the uterus and the atmosphere constitutes the most important propelling force at birth. After the rupture of the amnion membrane, the atmosphere sucks the child out from the uterus in which a high hydrostatic pressure rules.(2).

(2). Danelius followed the birth of a macerated fetus by serial x-ray pictures and was able to show the development of the typical moulding of the head with considerable elongation of the skull and the collection of a great amount of fluid in the scalp and in the subcutaneous tissue of the presented region. Danelius accepted his observations as proof of the working of pressure differences during delivery.

The suction power of the release principle also represents the main influence which damages the newborn.

2. *On the Normal Anatomy of the Cerebral Venous System*

In head presentations, the suction effect of the lower pressure disturbs primarily the large venous sinuses of the head, over-filling them with blood and impeding the circulation. This fact led us to investigate the venous system of the head, and especially of the brain.

In the newborn, the anterior part of the superior longitudinal sinus passes between the two frontal bones, and its medial and posterior sections between the parietal bones. The entire vessel is covered only by the soft and generally thin membrane of the sagittal suture. The straight sinus takes the blood collected in the Galenic vein and leads it into the jugular vein. In the newborn, the straight sinus and the superior longitudinal sinus are fused together in the area of the occipital fontanel which, in the majority of cases, is directly exposed to the suction power of the release effect during delivery.(3).

(3). The superior longitudinal sinus and the straight sinus are exposed to the suction effect even more in premature than in full term babies: the scalp and fontanels, like all spaces between the bones, are relatively wider.

In this connection, the following tributaries of the Galenic vein (Fig. 1), are important. (A). The *internal occipital vein,* which originates in the pia of the median occipital surface of the cerebral hemispheres, and supplies functionally important regions of the cortex. (B). The *basal vein of Rosenthal,* which supplies parts of the brain stem and collects roots from the region of the substantia perforata anterior. There is no doubt, from a functional standpoint, that circulatory trouble in the supply area of this vessel are of the greatest importance. (C). The third branch of the vein of Galen, the *lateral ventricular vein* collects a number of small vessels which supply the white matter of the occipital lobe and the white matter of the hippocampal gyrus. (D). The fourth branch of the Galenic vein, the *internal cerebral vein,* forms the direct continuation of the central stem, situated in the axis of the ventricle system. The tributaries of these vessels are the following branches: (a). the *chorioideal vein* which emerges from the chorioideal plexus, (b). the system of the *terminal vein* with its anterior and posterior ramifications. In regard to traumatic lesions

at birth, it should be noted that the most important branches of the terminal vein join just under the terminal stria. The stem of the posterior terminal vein, a rather long vessel, lies close to the terminal stria in the furrow between the caudate nucleus and the thalamus. (c). the *vein of the septum pellucidum.* (d). The *posterior vein of the callosal body.* (e). *The superior medial cerebellar vein.* (f). *The inferior posterior cerebellar vein.* (g). The *hippocampal vein,* and (h). *the vein of the pineal body.*

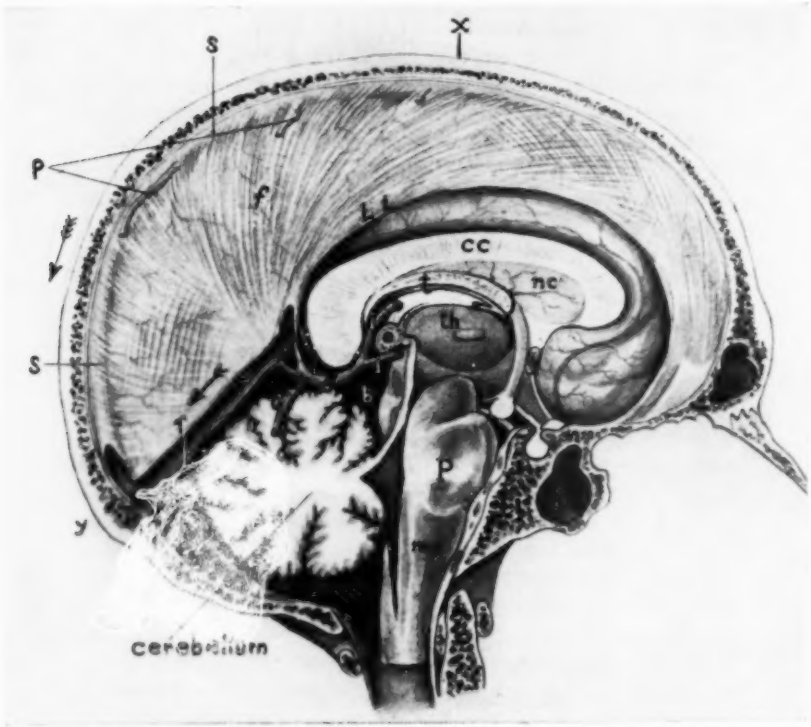

Fig. 1. The venous system of the head. (Combined from figs. published by Poirier and Testut). (*f*) falx; (*r*) straight sinus; (*g*) great vein of Galen; (*P*) pons; (*e*) pineal body; (*mo*) medulla oblongata; (*cc*) corpus callosum; (*nc*) caudate nucleus; (*th*) thalamus; (*s*) superior longitudinal sinus; (*li*) inferior longitudinal sinus; (*i*) internal cerebral vein; (*t*) terminal vein; (*p*) pial veins connected to the superior longitudinal sinus; (*x*) area of large fontanel in the newborn; (*y*) area of occipital fontanel in the newborn.

Differences in the effects which may be observed after injecting dyes into different sectors of the Galenic system are important to our understanding of the localization and extension of lesions in

Fig. 2. Venous supply of the basal ganglia shown by injection of dye directly into the internal cerebral vein. Note injection of thalamus, caudate nucleus and putamen. (*m*) cerebral white matter; (*cc*) corpus callosum; (*nc*) caudate nucleus; (*p*) putamen; (*pa*) pallidum; (*t*) thalamus.

birth injury. (Fig. 2; Fig. 3). If we inject the stem of the vein of Galen, we find most of the dye in the the white matter of the great hemispheres, while the thalamus and the striatum (caudate nucleus and putamen) show much less. However, if we inject

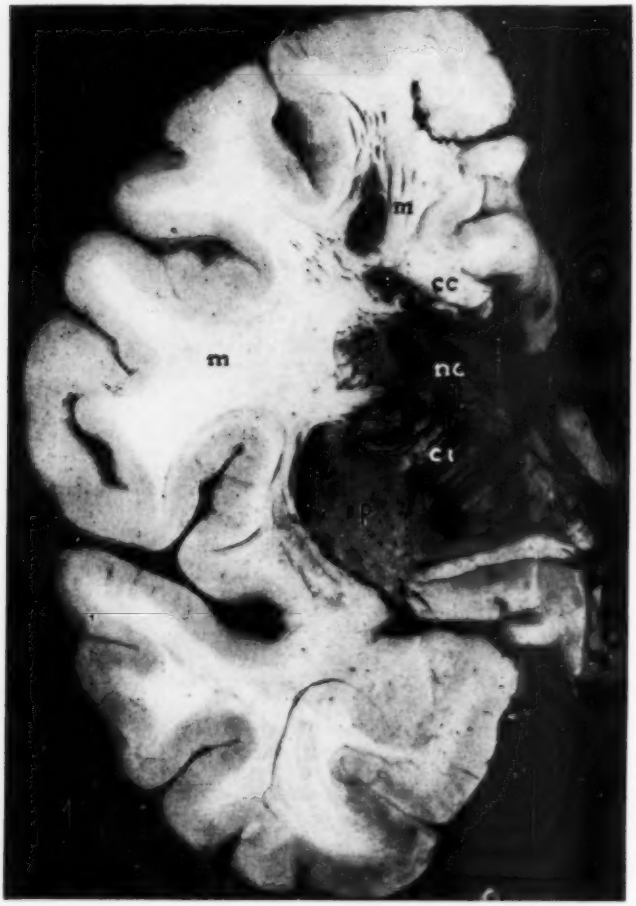

Fig. 3. Venous supply of the basal ganglia shown by injection of dye directly into the terminal vein. Extensive injection of the striate nucleus (caudatum and putamen) and of the pallidum. (*m*) cerebral white matter; (*cc*) corpus callosum; (*nc*) caudate nucleus; (*p*) putamen.

the dye directly into the internal cerebral vein, a very marked coloring of the thalamus, the caudate nucleus and the putamen appears. (Fig. 2). The direct injection of the terminal veins fills many vessels in both the caudate nucleus and the putamen. (Fig. 3).

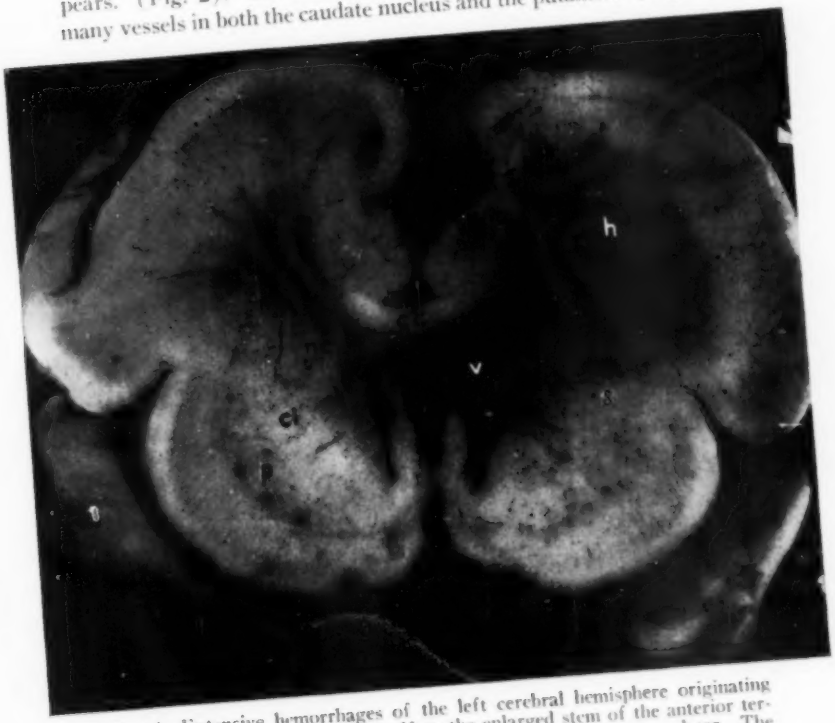

Fig. 4. Extensive hemorrhages of the left cerebral hemisphere originating from the anterior terminal vein. Note the enlarged stem of the anterior terminal vein filled with thrombotic masses situated below the ependyma. The ventricle is enlarged by hemorrhages originating from the subependymal branches of the terminal vein. Punctiform and strip-like hemorrhages form a massive focus in the white matter. Numerous minute hemorrhages affect the left striatum. Photo enlarged. Stillborn premature. (*h*) massive hemorrhage of the cerebral white matter produced by the confluence of capillary hemorrhages; (*v*) left ventricle filled by hemorrhage; (*s*) minute hemorrhages of the striatum; (*nc*) caudate nucleus; (*p*) putamen.

Of course, in all of these instances, the white matter of the hemispheres is also involved. However, this is not as marked as in cases where the dye was introduced into the Galenic vein itself. Undoubtedly, some differences exist between the venous supply of

the cerebral white matter and that of the large ganglia at the base of the brain; even though these regions are connected by the same vessels belonging to the vein of Galen.(4).

(4). The veins of the brain surface which are connected with the superior longitudinal sinus communicate with certain vessels which carry their blood to the cavernous sinus and the transverse sinus. The most important branch of these vessels, the *medial cerebral vein*, joins the superior longitudinal sinus directly to the venous sinuses at the base of the skull. Another quite large vein, also lying on the lateral surface of the occipital lobe, joins the posterior sector of the longitudinal sinus to the transverse sinus. A third important venous channel (the "grande veine anastomotique"), located on the lateral surface of the temporal lobe, connects the two large communicating veins with each other. No doubt, this net work of communicating veins plays a significant role in all cases in which the flow of blood, either in the longitudinal and transverse sinus system or in the cavernous sinus, is blocked unilaterally.

Bedford reported that a collateral circulation is rapidly established after the occlusion of the Galenic system. Schlesinger states that "the great vein of Galen partly drains the capillary network of the basal ganglia, and the chorioid plexus of the forebrain; although these structures can also be drained under certain circumstances by veins situated at the surface of the brain." "In the more extensive areas of the white matter, where the capillaries are scanty, large venous channels referred to as *intra-cerebral anastomotic veins* are present. They arise in the region of the angle of the lateral ventricle, from the anterior horn, backwards as far as the posterior horn, with their distribution following the arrangement of the callosal fibres. They establish the main intra-cerebral connections between the great vein of Galen and the veins at the surface of the brain."

"No anastomotic veins were found in the basal ganglia, where their absence may be related to the density and the richness of the capillaries and the larger vessels."

3. *Typical Traumatic Hemorrhages and Cerebral Softening in the Newborn*

At the peak of the labor pain, the difference in pressure is great enough not only to impede the blood flow in the superior longitudinal sinus and straight sinus but also to reverse the direction of the stream in the transverse sinus. This stopping of the circulation ("anoxia") may be sufficient to cause important brain damage. One effect is the accumulation of blood in the vein of Galen and in its tributaries. This short and relatively wide vessel shows changes in its shape and volume which are unique in pathology. Dilatations up to 3 cm. diameter filled with coagulated or liquid blood were repeatedly observed. (Turhan and Rössler). Thrombosis is not infrequent in this vein and is usually accompanied by thrombosis in the internal cerebral veins, the terminal veins, and the lateral ventricular vein. On the other hand, we have frequently seen cases in which only one of these last mentioned three vessels was thrombotic. *Hemorrhages* originating from the trunk and radicular

zone of the *terminal veins* are among the most frequent and, therefore, the most important lesions encountered in birth injury. They often destroy large parts of the caudate nucleus, and may infiltrate the frontal and fronto-parietal white matter. (Fig. 4; Fig. 5). Every stage is encountered from the smallest punctiform or linear lesion to large massive extravasations. *Bleedings* originating in the radicular zone *of the lateral ventricular vein* are equally typical of birth trauma and are found in the subependymal medullary substance of the occipital lobes.

Fig. 5. Extensive destruction of the subependymal matrix by bilateral hemorrhages of the terminal veins. (*m*) matrix infiltrated by hemorrhages; (*ci*) internal capsule; (*s*) septum pellucidum; (*v*) ventricle filled by hemorrhage.

Hemorrhages in branches of the terminal veins, the lateral vein of the ventricle, and the chorioideal vein occur solely in children injured during birth. We must emphasize that these bleedings do not arise from ruptures in the stems of larger vessels, but are the products of the confluence of countless minute hemorrhages of diapedetic nature, occurring in the terminal area of the vessel, or originating in the vasa vasorum in the walls of the

larger vessels. In many cases, these lesions may be discerned by the naked eye; sometimes, only microscopically. Macroscopic and microscopic bleedings in the thalamus, hypothalamic area, the midbrain, pons, medulla oblongata, and in the cerebellum are very common.

Bilateral involvement is one of the most interesting characteristics of intracerebral circulatory disturbances at birth. Bilateral vena terminalis bleedings, (Fig. 5) and bilateral hemorrhages in the domain of the chorioideal vein or of the lateral ventricular vein are in various combinations, very frequent. It is noteworthy that the intracerebral bleeding generally appears to be greater in the hemisphere which is presented at birth. If intracerebral bleeding occurs unilaterally, we usually find it located on the side that was presented at delivery.

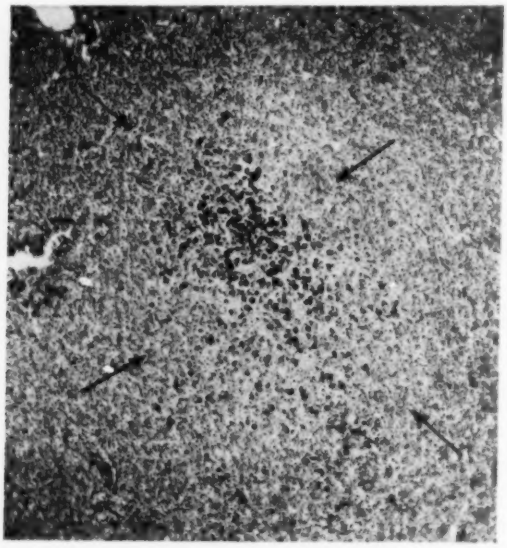

Fig. 6. Minute softening focus located in the putamen in a 5-week-old child. Stained with scarlet red. (A. 1419/25 Frankfurt-Main)

Sequelae of the circulatory disturbances may frequently be observed in older children. We often see, even with the naked eye, typical localized scars associated with hemosiderin deposits. Extended masses of pigmented coagula, adhering to the dura or to

the pia and infiltrated by granulation tissue, sometimes denote the typical condition of a chronic internal pachymeningitis. Thrombi in the large sinus of the skull slowly transform themselves into a pigmented connective tisue. The thrombotic veins of the pia, in the walls of the ventricles, in the falx, and in the tentorium may be preserved as rustbrown strips, visible many years after birth. We have observed the recanalization of veins which were occluded by thrombi during birth.

As a consequence of the circulatory disturbances in the whole central nervous system and especially in the brain, typical *softening* appears in many cases. This may be characterized by: (1) disintegration and dissolution of the affected brain substance; (2) rarefaction of the nervous tissue, in which its basic structure, even if only in its essential features, is preserved.

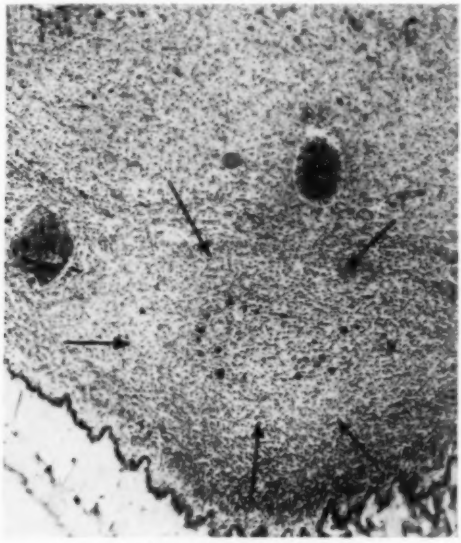

FIG. 7. Minute scar with cells containing hemosiderin, located in the subependymal area of the caudate nucleus. Stained with scarlet red. A 3-month-old child. (A. 377/26, Frankfurt-Main)

These softenings present themselves either as circumscribed or as diffusely extended foci. The foci of dissolution in the newborn are mainly around the lateral ventricles, in the frontal,

frontoparietal and occipital white matter. They arise from circulatory disturbances in the supply area of the terminal vein and the lateral ventricular vein. Small foci of dissolution in the newborn are extremely frequent, almost without exception present in children who have died from two to thirty days after birth, and are usually visible to the naked eye. Diffuse dissolution destroys primarily the white matter of the cerebral hemispheres. Thus, central porencephalic defects appear, the development of which can be followed from its beginning in the first days after birth to the final scars. We found that in a preponderant number of cases "congenital" porencephaly constitutes typical sequelae of the traumatic lesion at birth. Localized and diffuse rarefaction processes in the newborn also affect primarily the white matter of

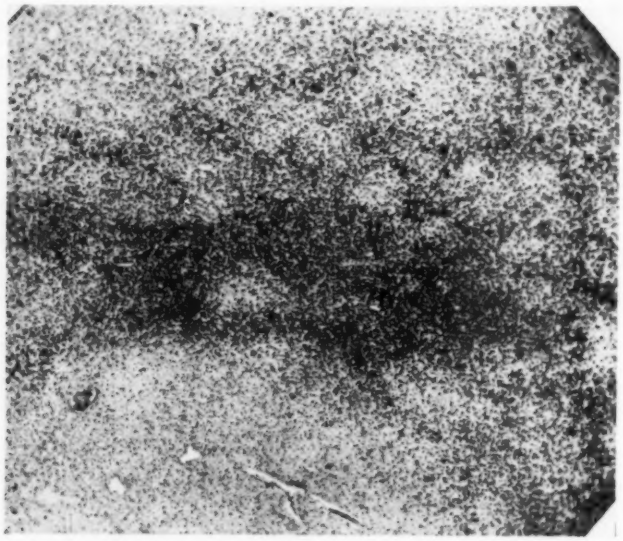

Fig. 8. A net-like pattern of rarefaction in the nervous tissue of the thalamus in a 30-day-old premature. This child weighed 1780 gms. at birth. At autopsy it showed typical birth traumatic softening foci of the white matter of the cerebral hemispheres. (A. 701/26, Frankfurt-Main). Stained with scarlet red.

the cerebral hemispheres, which is the area of supply of the terminal veins and of the lateral veins of the ventricles. The severest alteration of this type finally produces conditions which

are often designated as congenital lobar or diffuse sclerosis of the brain. We have frequently found recent and old foci of softening of various sizes together with their scars in the midbrain, pons, medulla oblongata and cerebellum.

Diffuse and circumscribed softenings also occur frequently in the large ganglia at the base of the brain. We had the opportunity to investigate rarefaction processes in the striatum and in the thalamus from their beginnings at birth to their full development into typical *status marmoratus.* (5). (Figs. 6 to 12).

(5). *Wald,* an associate of the author's, investigated one hundred and twenty-seven brains of stillborn children and of infants who died following birth. The oldest child was 8 months old. We found fifty-nine cases in which birth traumatic lesions of the white matter and basal ganglia were simultaneously present, while in twenty-two cases only the white matter showed damage.

III. *Development of Status Marmoratus as a Result of Circulatory Disturbances at Birth.*

Fully developed status marmoratus of the striatum and thalamus constitutes a scarring secondary to a diffuse rarefaction process.

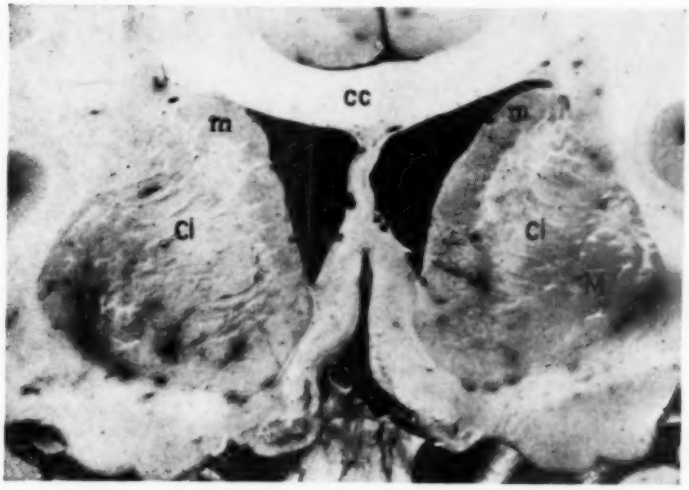

Fig. 9. Bilateral status marmoratus of the striatum in a 15-year-old girl with Little's disease. (A. 1619/31, Frankfurt-Main). The white spots and strips of the hypermyelinated net-like scars were clearly visible microscopically. m—hypermyelinated spots and strips of the caudate nucleus; M—hypermyelinated spots and strips of the putamen; ci—capsula interna; cc—corpus callosum.

Status marmoratus is characterized by parenchymal defects, fiber networks of mesenchymal and glial origin, as well as of bundles of irregular nerve fibrils arising from useless regeneration. (Fig. 8-12). The size of the diseased tissue is markedly diminished, while its consistency is increased. The cut surface shows an irregular entanglement of white myelinated fibers which may fuse and produce irregular conglomerations. The larger the lesion, the more varied is the pattern. We repeatedly found a whirl-like design in the thalamus. The tendency of the infiltration to limit itself to structural ("topistic") units is especially striking in the putamen. Status marmoratus often seems symmetrical, affecting both caudate nuclei, putamina or thalami; but the lesions never appear identical on both sides.

Fig. 10. Bilatral status marmoratus of the thalamus in a 15-year-old girl. m—hypermyelinated spots and strips of the thalamus; th—thalamus; ci—capsula interna; r—nucleus ruber; P—pons; cc—corpus callosum.

There are cases in which the structural change of the cut surface may be recognized without any difficulty, even in the fresh brain. (Fig. 9, 10, 11). In other instances, delicate cloudy spots are perceptible only in stained sections, with the help of a magnifying glass. The more expertly we know the macroscopic appearance of the great basal ganglia, the more we are able to

detect irregular strips and spots, which on microscopic examinations prove to be entanglements of myelinated nerve fibrils in scars. *The classic picture of "status marmoratus" described by Anton and by C. Vogt represents the most marked degree of a lesion which often produces only small foci without any resemblance to a polished piece of marble.*

The following peculiarities may be observed in brains in which accentuated "status marmoratus" of the basal ganglia is present: multiple parenchymal defects, scars containing glial fibers, and foci of entanglements of myelinated or naked, irregular nerve fibrils. Along side of areas of hyper-myelinization, there are spots characterized by a lack of myelinated nerve fibrils. In severe status marmoratus cases, we usually find shrinking of the white matter, especially in the frontal parts of the hemispheres. (Fig. 11). These areas feel harder than normal and their myelin content appears diminished. We often encounter a combination of status marmoratus in the large basal ganglia and sclerotic microgyria or other cortical deformity caused by scarification. (Fig. 11). The similarity of cortical scars and foci to status marmoratus of the basal ganglia is remarkable. Large scars and deformities of the basal ganglia frequently occur on the same side which is affected by status marmoratus or in the other hemisphere. We have observed status marmoratus in cases where the white matter of the hemispheres showed cavities typical of porencephaly.

Anton and C. and O. Vogt defined "status marmoratus" of the basal ganglia as a condition which appears in the brain without any other damage. The more new cases observed, however, the more evident it became that "status marmoratus" of the basal ganglia represents a condition which accompanies a wide-spread damage of the entire brain. Thus, *status marmoratus should not be considered a selective disease of the basal ganglia. Rather, it is a striking sign of damage to the entire brain in multiple foci.*

All the above mentioned changes of the brains which show "status marmoratus" of the large basal ganglia may be caused by circulatory disturbances from birth trauma. We have performed many autopsies in which such diffuse or localized damages to the basal ganglia were recognized in the first days or weeks after birth. In the same cases, we usually found those lesions of the cerebral white matter and cortex which, when scarified, characterize "status marmoratus." In instances in which considerable diffuse damage

Fig. 11. Bilateral status marmoratus of the striatum in a 9-year-old boy with idiocy and Little's disease. The net-like arrangement of hypermyelinated scars was visible macroscopically. Shrinking of the white matter in both hemispheres caused by diffuse scarification. Extendive porencephalic defects of the cerebral hemispheres were also present. (A. 1193/26, Frankfurt-Main).
m—hypermyelinated spots and strips of the caudate nucleus; M—hypermyelinated spots and strips of the putamen; w—cicatricial contraction (sclerosis) of the cerebral white matter; s—sclerotic mocrogyria; cc—atrophy of the corpus callosum.

to the basal ganglia was present, hemorrhagic destruction of the superficial parts of the caudate nucleus and of the thalamus were common. In children who died shortly after birth, the origin of this damage from venous bleedings was manifest.

The author collected about twenty-five cases in which "status marmoratus" of the ganglia at the base of the brain was present in a fully developed degree. In all of these instances the origin from birth lesions was clear.

Fig. 12. Status marmoratus of the right striatum. (Weigert's myelin stain). nc—caudate nucleus displaying many spots and strips of irregularly myelinated nerve fibrils; p—putamen displaying many spots and strips of irregularly myelinated nerve fibrils; ci—capsula interna.

During intra-uterine life, as well as in the early stages after birth, certain diseases of infectious or toxic origin (Hollinger, 1933, Wolf, Cowen and Paige, 1939) or secondary circulatory disturbances of the brain, may involve regions typically affected by "status marmoratus." It would, therefore, seem permissible to assume the existence of "status marmoratus" caused by infection

or toxic influences. We must emphasize, however, that as yet no instance of such causation has been reported that could be accepted as established.

In view of the observations discussed above, the most common pathogenesis of status marmoratus may be described as follows:

The differences in pressure which act on the head of the infant during delivery cause the interruption of the intra-cerebral venous blood flow. A softening process in many parts of the brain ensues. If the child survives the damage, scarification leads to development of foci in the large strial or thalamic areas with the typical aspect of status marmoratus.

The development of myelinated nerve fibrils which bring about the striking appearance of status marmoratus in the basal ganglia, results from the abundant, very irregular, regenerative proliferation of nerve fibers which were damaged by the initial lesion. Thus, similar phenomena may develop in every part of the central nervous system (basal ganglia, cerebral white matter, cerebral cortex, etc.) in which abundant regenerative proliferation of nerve fibers occurs. The changes will appear to be particularly conspicuous in parts of the nervous system, which normally are of a gray color, due to the limited amount of myelinated structures.

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MYASTHENIA GRAVIS: FOUR CASES. M. Latorre and R. Reynoso. (Rev. chilèna pediat., 26:471-476, Nov.-Dec. 1955).

Myasthenia gravis is very rare in children. Four cases were observed during the last six years in the Luis Calvo Mackenna Hospital of Santiago, Chile. The patients were three boys between the ages of 7 and 14 years and a 10-year-old girl. The symptoms in all patients were similar, consisting of extreme fatigability, especially in the morning, palpebral ptosis, lowering of the angles of the lips, difficulty in deglutition and in speech, strabismus, and facial paralysis. The symptoms were more severe in the course of infections. The prostigmine test was positive in all. Mental and sensory reactions were normal. A roentgen examination of the thorax for shadows of persistent thymus was made in two cases, with a positive result in one case and a negative result in the other. The treatment consisted of prostigmine by mouth in doses of 15 mg. every six hours up to a total daily dose of 60 mg. This dose was maintained for a certain time and then it was progressively diminished until its discontinuation in, as a rule, about a year. The symptoms were controlled in all patients but one. They reappeared in this patient two years after the cure. They were due to strenuous physical effort during sport and were permanently controlled by administration of prostigmine on the above schedule for a short time. One of the patients, after having experienced great improvement, developed acute respiratory disorders that did not respond to treatment. He died. Autopsy showed bronchopneumonia of the lower lobe of the left lung and diffuse hyperemia and hyperplasia of the thymus (thymus weighed 60 gm.). The other three patients were normal at the time of writing, from four to six years after the cure of the disease.—*J. A. M. A.*

MODERN TRENDS IN THE TREATMENT OF APPENDICITIS*

DANIEL H. MANFREDI, M.D., D.A.B.S.
New York.

The appendix or "worm of the bowel" as described in the Coptic era has been crudely represented attached to bowel in the Greek mythology and pottery. Aristotle and Galen were ignorant of its existance since their anatomical studies were confined to lower mamallian orders.

In 30 A.D., Aretus described a patient who recovered from an appendiceal abscess after simple drainage of the abdominal wall. Vesalius described the organ but never knew of its pathology. In 1753, Heister described a post-mortum appearance of a gangrenous and perforated appendix. In 1759, Mestivier was the first modern surgeon to drain an appendiceal abscess. In 1839, Bright and Addison accurately described the symptomatology of an appendiceal abscess without realizing its cause. Fitz in 1880 coined the term "appendicitis". In 1885, Mickulitz successfully operated upon a patient with an appendiceal abscess, and in 1887, Morton first successfully removed an appendix in a case of appendicitis.

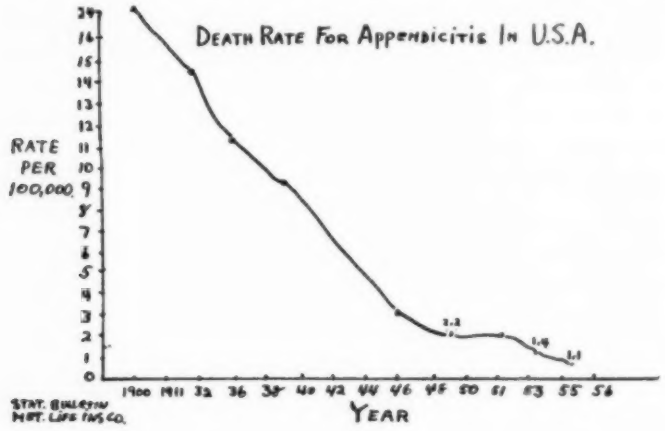
Since those early days of surgery, the management of appendicitis has undergone many changes. There is almost universal agreement that in the early stages of appendicitis before perforation or abscess occurs, the treatment should be immediate appendectomy. There is no drug or medical treatment for appendicitis. The antibiotics will not cure appendicitis. This was convincingly proven to us in the past month by two patients being treated for otitis media with large doses of antibiotics who developed acute suppurative appendicitis.

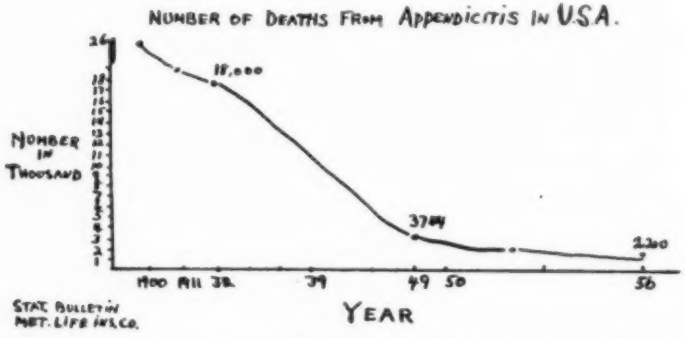
In the late stages of appendicitis, especially with abscess formation, the treatment becomes less standardized. As most of the deaths in appendicitis occur following the onset of the late stages, one of the urgent necessities for which we should strive, is the enlightenment of the family doctor, and especially of the laity, to the early symptoms of the disease. In addition to this, they should be warned against procrastination and laxatives.

In 1956, 2200 Americans died of appendicitis who could have been saved. In the year 1900 the overall mortality rate for append-

* From the Knickerbocker and Misericordia Hospitals, New York, N. Y.

icitis was 24 per cent in the U. S. A. In the year 1930, the death rate was 15.2 per 100,000; in 1948, it had dropped to a low of 2.9 per 100,000; and in the year 1956, the rate had fallen to 1.4 per 100,000. Certainly we should rejoice in this improvement as it is


DEATH RATE FOR APPENDICITIS IN U.S.A.
RATE PER 100,000
1900 1911 32 36 38 40 42 44 46 48 50 51 53 55 56
YEAR
STAT. BULLETIN MET. LIFE INS. CO.



a great credit to the surgical profession. However, we must constantly be aware of the fact that it is a disease in which a zero mortality can be expected if a concerted effort is made by all.

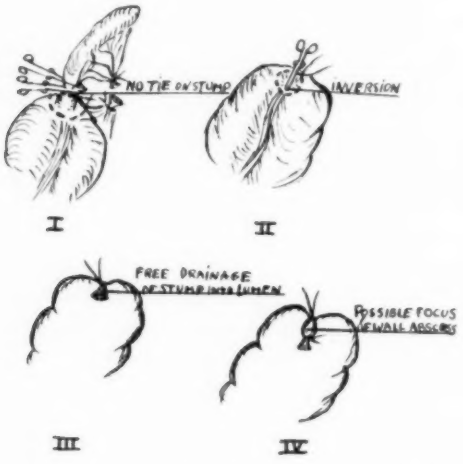
Dr. Ochsner, before the advent of the antibiotics, observed that the mortality in appendicitis was high if the treatment of immediate operation were performed after the fourth day of the onset of the illness. He advised, at that time, a careful watchful waiting

and then drainage when fluctuation occurred either by rectum or the abdominal route. His method had certain disadvantages. It could not be applied to children as localization was less satisfactory since the short and meagre omentum cannot surround the appendix adequately. If the operation became urgent, the danger of opening an abscess was the dissemination of infection. At Bellevue Hospital, prior to the advent of the antibiotics, appendiceal abscesses in children were treated by a partial waiting period. Many of these patients would enter the hospital three to five days after the onset of their illness in a state of marked dehydration, electrolyte imbalance, low protein levels and with distension. Delay in operating was governed by the time required to bring the patient into good physiological, nutritional and electrolyte balance. In the abscess cases, drainage with as little manipulation as possible was performed, with removal of the appendix when it appeared feasible and if we were sure that it would not disseminate the peritonitis. If it were not advantageous to remove it, simple drainage with partial closure of the peritoneal cavity was performed. The abdominal wound from the peritoneum outward was packed with vasoline gauze. We always felt that the peritoneal cavity could handle infection much better than the abdominal wall with its lowered resistance when exposed to infection.

With the advent of the sulfa drugs, the usage of intraperitoneal sulfathiazole, placed into an abscess cavity was considered a step forward toward lowering the mortality rate. After World War Two, the antibiotic era was in full swing and early administration of antibiotics was added to the armamentarium of early restoration of physiological balance of electrolytes, proteins and fluids, coupled with gastro-intestinal suction to place the bowel at rest. A bolder approach was justified. The abdominal wall now could handle contaminating infection when assisted by the aforementioned adjuvants. Our period of waiting has been cut down to a minimum, and we feel that this serious and dangerous source of infection should be removed earlier. With this procedure, our mortality rate has dropped to one per cent. At present, we advise early emergency appendectomy in all cases diagnosed as acute appendicitis or strongly suspected as such. It is always better to operate than risk the possibility of a perforation. In cases of 72 hours duration or longer in which there is evidence of a spreading peritonitis with generalized toxicity and no localizing signs, we pre-

scribe antibiotics, intravenous feedings of glucose and electrolytes, decompression by intraluminary tubing, Wangensteen suction, semi-Fowler position and nothing per oram. An appendectomy or drainage is performed at a later date.

In doing appendectomies on our acute cases, we favor the McBurney incision because of its direct approach upon the appendix and also because of the low percentage of post-operative hernias that have been noted, following its use. We routinely invert the stump of the appendix with intestinal suture on an atraumatic needle. The stump is treated with alcohol and is left open



METHOD OF INVERTING STUMP TO PREVENT WALL ABSCESS

without a ligature. In this manner we avoid an intramural abscess. The technique is carried out with a small clamp which acts as a temporary ligature and helps to invert the base of the appendix. If the base of the appendix and cecum are involved in the inflammatory process and we suspect that the sutures are apt to tear through, the stump is ligated with chromic catgut without inverting it. These cases are drained with a rubber tissue drain down to the pericecal area. Occasionally, a retrocecal appendicitis may contaminate the retroperitoneal fat which is susceptible to infection. Postoperatively we continue to administer antibiotics

because they reduce postoperative infections such as subphrenic abscess and pylephlebitis. These new drugs have extended the possibilities of surgery but they have not lessened the basic problems.

In summary we may state that the remarkable drop in mortality from acute appendicitis has been one of the milestones in the progress of modern surgery. Among the attributes that go to make up this phenomena are included the advent of the newer drugs, improvement in the administration of anesthesia, better preoperative preparation of the patients with fluids, electrolytes and intravenous feedings. Also the earlier diagnosis and operating with more scientific postoperative attention. There still were two thousand deaths from appendicitis in the U. S. A. last year. This figure can be cut down considerably if the lay public and family doctor are constantly enlightened as to initial symptoms and signs so that an early operation can be performed. This would be a great step forward since most of the deaths that occurred were in the neglected cases. And finally, we should abolish that mythical credo that appendicitis can be cured with antibiotics alone.

THE PROGNOSTIC SIGNIFICANCE OF NEONATAL CONVULSIONS. (Arch. Dis. Childhood, 29:342-345, Aug. 1954). The causes of convulsions during the first few days of life include intracranial damage, anoxia, congenital malformations, tetany, and infections. It is not always possible to determine the cause, but the prognosis is of great importance. Case histories of all infants recorded as having convulsions or muscular twitchings were studied in a Sheffield hospital over a five year period. There were 46 cases in a total of 8,679 deliveries (*.2%). Eighteen infants died during the first 13 days of life, 28 were discharged from hospital, and 27 have been followed up. Five of the 27 were severely retarded mentally; spastic quadriplegia was present in one of these, and further convulsions occurred in 2; a 6th child was blind in the left eye. Convulsions (or muscular twitchings) occurring in the newborn period were associated with birth trauma, asphyxia, or cyanotic attacks in 35 cases. It is clear from the present series that convulsions and muscular twitchings are of serious significance in the newborn period.—*J.A.M.A.*

Obituary

HAROLD RUCKMAN MIXSELL, M.D.
1896 - 1958

The Publisher and Editors of ARCHIVES OF PEDIATRICS deeply regret the death, on February 13, of their esteemed colleague and member of the Editorial Board, Dr. Harold Ruckman Mixsell. Dr. Mixsell served as Editor-in-Chief of this journal for approximately 20 years. A number of his scientific articles on pediatric subjects were contributed to other medical journals.

He was born in Easton, Pa., and received his A.B. Degree from Princeton in 1906, and his M.D. from the College of Physicians and Surgeons of Columbia University in 1910. For many years he engaged in a large pediatric practice in New York City, and was still active up to his sudden death.

Dr. Mixsell had a wide variety of medical interests. In addition to his practice and his editorial duties on the ARCHIVES, he found the time to serve as pediatrician to the Woman's Hospital of New York City where he supervised the care of the newborn from 1925 to 1948. He was also Consulting Pediatrician to St. Luke's, Beekman Downtown, Knickerbocker and Glen Cove Community Hospitals.

He was a Trustee of the New York Academy of Medicine, and Chairman of their Medical Information Committee. His other memberships included the American Medical Association, the American Academy of Pediatrics and the New York County Medical Society. At one time he served as a Faculty Member of the New York Postgraduate Hospital.

Dr. Mixsell's personality and warmth won him a host of friends. He will be sorely missed by the ARCHIVES.

PEDIATRICS AT THE TURN OF THE CENTURY

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

CONGENITAL LARYNGEAL STRIDOR APPARENTLY DUE TO AN ENLARGED THYMUS GLAND*

A. W. MYERS, M.D.
Milwaukee, Wis.

In one of their papers on the subject Thomson and Turner give in a single paragraph so clear a picture of a typical case of congenital stridor that it is well worth quoting: "The infant who appears normal in other respects, is noticed shortly after birth to have noisy breathing. The noise consists of a croaking sound accompanying inspiration, which rises to a high-pitched crow when a longer or more vigorous breath is taken. Expiration may be accompanied by a short croak when the stridor is loud, but at other times it is noiseless. Even in the most severe cases there are occasional brief intervals, during which there is no sound audible; but, with this exception, the stridor goes on constantly while the child is awake, and sometimes even when he is asleep. Any emotional excitement, or any physical cause of deeper breathing, such as exposure to colder air or the exertion of sucking, is apt to intensify the sound. The child's power of crying and coughing is unaffected. The breathing, though noisy, is not accompanied by the slightest distress, and there is no cyanosis. There is, however, always marked inspiratory indrawing of the thoracic and abdominal walls, except in the mildest cases."

While the clinical features of congenital laryngeal stridor are well defined, there is still much disagreement as to the manner of its causation, and the definition which Rabé gives in his recent excellent article on the subject seems to be hardly justifiable. He defines the condition as a manner of respiration characterized by an inspiratory noise, sonorous and continuous, appearing imme-

* Read before the Chicago Pediatric Society on March 17, 1908.
** Reprinted from ARCHIVES OF PEDIATRICS, 25:607-616, August 1908.

diately after birth, or several days after, and *connected with the existence of congenital malformations of the larynx*. These malformations of the larynx he would divide into two types following Variot's classification. In the first group are those cases in which the deformity is very marked, with the epiglottis folded on itself into a narrow gutter, and the aryteno-epiglottic folds so soft and flabby as to approach or meet in the middle line, leaving only a narrow chink between. There is thus formed a vestibule, which sinks in and vibrates under the influence of the current of inspired air. The second group comprises those in which the deformity is less marked and is limited to the epiglottis, which is rolled on itself in the same manner as in the first group, but to a lesser degree. By reason of this rolling the aryteno-epiglottic folds are brought into contact, but only in their anterior thirds. In this type the stridor results chiefly from the vibration of the epiglottis. No provision is made in this classification for those cases in which the clinical picture is present in its entirety, and in which no laryngeal abnormality can be demonstrated.

Thomson and Logan Turner are inclined to look upon these laryngeal or vestibular deformities, which unquestionably are present very frequently, as acquired factors, the result of poorly-coordinated and spasmodic inspiratory movements acting upon the soft and pliable tissues of the infantile larynx. From this point of view the primary defect is in an arrest of development of the cortical center for respiration, so that a type of incoordinated respiration is produced, which may be compared with stammering.

The other important theory as to the etiology is that of tracheal compression, most often by an enlarged thymus gland. This was advocated by Kopp nearly eighty years ago, and in recent times by Avellis and Hochsinger. Avellis based his belief in the thymic origin of congenital stridor on the similarity between the symptoms of this condition and those seen in some cases of hypertrophied thymus, on the clinical course of the process which suggests its connection with the thymus, and on the results of treatment. He did not discover any physical signs of enlarged thymus in the four cases he reported. He mentioned the occurrence of congenital stridor in several members of the same family, but no attempt was made to draw any conclusions from this fact, which has been mentioned by a number of other observers.

Hochsinger studied the size of the thymus by means of x-ray plates in 26 cases of congenital stridor, and found definite enlargement in every case except one. He also found the thymus enlarged in 20 out of 32 other young children suffering with rickets, and the frequency with which he demonstrated thymic hypertrophy seems to have militated against the attachment of much weight to his report.

On account of the diversity of opinion on the question of etiology, the report of the following case would seem to be of interest, because it shows the coexistence of congenital stridor with thymic enlargement, and the disappearance of the stridor when the thymus was reduced in size by treatment.

M. B., female, the only child of young and healthy parents, was born July 21, 1907, a forceps delivery terminating a comparatively short labor. There was no asphyxia and no cyanosis, but it was noticed that the respiratory rate was very rapid from the time of birth. When the baby was a week old the inspirations were observed to be labored in character, and a crowing sound began to be heard with inspiration. The stridor gradually became more marked, the inspiratory difficulty steadily increased, and by the time the baby was three or four weeks old the bulging forward of the sternum with inspiration appeared. The child was always breast fed, there was never any difficulty in swallowing, there was no vomiting, the stools were always good, and gain in weight was steady throughout. There was no cough, and never any cyanosis.

When the child was seen for the first time, on September 13, at the age of about eight weeks, she was found to be a normally developed and well nourished child, weighing 9 pounds, an increase of 2 pounds over her birth weight. Her skin and mucous membranes and finger nails were of good color. Her tongue was clean and not enlarged, her pharynx was normal, nasal respiration was free, and no adenoids could be found in the nasopharynx. Her abdomen was normal, and the superficial lymphatic glands were not enlarged.

On inspection of the chest one was impressed by the labored character of the inspirations. These were accompanied by so marked a recession of the suprasternal notch and the epigastrium that the upper and lower ends of the flexible sternum were approximated, producing a remarkable forward bowing of

this bone. At times it seemed to bend almost into a semicircle, having the line of its convexity in the long axis of the body. With this there was an indrawing of the intercostal spaces and a slight depression laterally on each side of the sternum. The crowing sound which accompanied the inspirations was very plainly heard even when the child was in an adjoining room with the doors closed. The crowing and the labored inspiration were almost continuous, although when the baby was very quiet she would sometimes breathe softly and naturally for a minute or two. The mother of the patient stated that when the infant was in a deep sleep the quiet breathing would sometimes last for nearly half an hour, but never longer. When the child cried or became excited from any cause the stridor and labored breathing became more marked, but at no time did the voice sound hoarse or harsh or in any way abnormal. Even when the breathing was most labored the expression on the child's face was placid and contented. Expiration was normal.

Behind the upper part of the sternum a mass could be easily outlined by percussion. It was an irregular quadrilateral in shape, the upper border extending from the right sternoclavicular articulation 2.5 cm. to the left, into the first intercostal space on the left side. The right border followed the right edge of the sternum a distance of 3 cm. down to the third interspace. The lower border at this level measured 1.5 cm. transversely, and the left border was formed by an oblique line connecting its termination with the left end of the upper margin. In addition to the dull percussion note over this area the feeling of resistance to the percussing finger was very definitely localized and helped to define sharply the outline. By introducing the tip of the finger deep into the suprasternal notch it seemed as though a mass could be felt indistinctly with expiration, but this procedure caused so much respiratory disturbance that a definite decision was not reached.

The lungs seemed fully expanded and everywhere resonant, except over the right apex posteriorly where the percussion note was slightly impaired. There was also a small area of dullness in the interscapular region just to the right of the spine. The breath sounds were harsh and rhonchi were heard everywhere.

The child seemed to prefer to lie on her left side and the stridor was increased by placing her on her back, and still further increased when her head was overextended or overflexed. At

times, when the stridor was marked, raising her from a lying to a sitting position apparently added to her comfort, but did not cause a disappearance of the crowing. These last-named features are the only ones that would enable one to differentiate clinically such a case as this from those cases in which the larynx is markedly deformed.

A laryngoscopic examination was made on the day the child was seen for the first time, and a normal infantile larynx was found. A subsequent examination was made several weeks later, and these findings were confirmed.

The normal evolution of a typical case of congenital laryngeal stridor is interesting in its course. The characteristic inspiratory noise, which usually appears at birth, or in the first few days of life, gradually increases in intensity during the second and third months; it then remains stationary until about the eighth month. After this time it gradually decreases and disappears by the end of the second year. In one of Variot's cases the stridor had disappeared by the end of the eighth month, and even earlier cures have been reported, while in one of Comby's cases the condition persisted into the third year, and in Perrucker's patient operation was performed for the relief of the symptoms at the age of two and one-half years. In most cases there is no interference with the general growth and development.

While the outcome is almost invariably good, in spite of the alarming character of the symptoms, it is well to remember that certain complications may seriously affect the prognosis. In cases of laryngeal stridor the occurrence of attacks of laryngitis, bronchitis, bronchopneumonia and lobar pneumonia is especially serious. Ten autopsies have been made on cases where this condition was present during life, and in all but two death was due to disease of the respiratory tract. Four deaths were caused by bronchopneumonia, and one each by lobar pneumonia, laryngeal diphtheria, simple laryngitis and pulmonary tuberculosis. Of the other two deaths one was due to scarlet fever and one to convulsions. In this last case no macroscopic or microscopic changes could be found, except a moderate deformity of the larynx.

When laryngeal abnormalities are present and no changes in the thymus can be found, it may be necessary to do very little, except to await the natural evolution of the process, using such measures as may be needed to maintain the general health of the patient at its highest level and carefully treating the slightest

derangements of the respiratory tract to prevent the development of more serious pathological conditions, although attacks of dyspnea of great severity might require intubation or tracheotomy.

But when there is an enlargement of the thymus, demonstrable either by physical signs or by x-ray examination, or strongly suggested by the symptoms, it seems hardly justifiable to follow a course of "masterly inactivity," since the tragic possibilities of this condition are so well known.

Operative intervention in enlargement of the thymus was practiced for the first time by Rehn, in the case reported by Siegel in 1896. The patient was a boy two and one-half years of age, who was well until a difficulty in breathing was noticed five weeks before admission to the hospital. This increased steadily, and at the time of admission the dyspnea was so severe that a tracheotomy was performed at once. No relief was afforded until a long canula was introduced, which reached almost to the bifurcation. Every attempt to replace the long canula by a shorter one was followed by a return of the symptoms. An enlarged thymus was suspected, although no physical signs of it could be detected. A median incision was made, and a mass of thymus gland the size of a hazel-nut was found presenting with expiration, while with inspiration it sank back into the mediastinum. The thymus was drawn out as far as possible and stitched to the fascia covering the sternum. The wound was then closed. Recovery was complete.

Rehn's second case was one of congenital inspiratory stridor, with cyanosis added to the typical symptoms. In this case a small rounded tumor could be noticed in the jugulum during expiration. Operation was performed at the age of four months. A median incision was made and the capsule of the gland divided. As the gland substance tore very readily and disappeared deep in the mediastinum with each inspiration, it was necessary to make use of each expiration to remove a piece until its size was reduced to such an extent that it could be lifted up by a dull curette. Rehn recommends that the capsule be sewed into the wound so that the space left may be drained. He thinks that complete removal of the gland is hardly possible and that resection of the sternum is not necessary in ordinary cases. If it seems desirable the operation may be done without anesthesia.

In 1897, König operated upon a baby nine weeks of age, which had suffered from inspiratory dyspnea and cyanosis from the sec-

ond week of life. A soft, rounded tumor appeared in the jugulum with expiration. After drawing the gland up into the incision the capsule was split and the greater portion of the gland removed. The remaining stump of the gland was fixed by stitching the capsule to the sternum and the tendinous attachment of the sternomastoid. Recovery was good, but shortly after the operation the child developed a severe rachitis and did not walk until four and a half years of age.

König's second patient was in the first year of life, and presented symptoms similar to those just mentioned. The stridor was both inspiratory and expiratory, and in this case also there was a palpable thymus. König removed the right lobe of the thymus and fixed the left lobe to the fascia, and also did a deep tracheotomy, but the symptoms persisted. At a second operation, the remainder of the thymus was removed and the opening of the thorax enlarged by the resection of a portion of the manubrium. Complete recovery followed.

Perrucker's patient was a child of two and a half years, but differed from Rehn's first case in that he had presented from birth the typical picture of congenital laryngeal stridor, with the addition of extreme hoarseness and a weakened voice. No physical signs of an enlarged thymus were present, except that auscultation revealed a murmur of stenosis equally clear on both sides. Operation showed the thymus to be greatly enlarged, and the moment it was drawn forward the stridor disappeared. The gland was removed and recovery was complete, but the hoarseness persisted, although the larynx was normal.

Aphonia was also present in Ehrhart's case, that of a girl two years of age, and the other symptoms resembled closely those of Perrucker's patient, but, instead of being congenital, the condition developed acutely with fever two months before coming under observation. The larynx was normal and no physical signs of enlarged thymus could be detected. Intubation gave no relief. A median incision was made, and as the tissues were dividied an enlarged thymus was seen presenting in the jugulum with each expiration. Its enucleation was not especially difficult and the relief obtained was prompt and permanent.

In J. L. Morse's case dyspnea existed from birth, although its character differed from that of the typical case of congenital stridor. Both inspiration and expiration were noisy and difficult, especially the latter. The larynx was normal and the thymus

could not be felt. No area of dullness could be made out under the manubrium. A diagnosis of hypertrophy of the thymus was made, but the consent of the parents to an operation could not be obtained until after a long delay, during which the child's general condition grew steadily worse. At the age of eleven weeks the baby was operated upon by Dr. F. T. Murphy, and the thymus was found to turn back against the trachea in inspiration. Upon drawing it forward the dyspnea, both inspiratory and expiratory, was markedly relieved. When the gland was allowed to drop back the dyspnea recurred. The thymus was drawn forward and anchored and a part of the manubrium was cut away to enlarge the opening. Although the respirations became less labored the child did not do well, and death occurred sixteen days after the operation. Postmortem, the gland was found anchored in its new position, where it seemed to exert no pressure upon the trachea.

The most recent operation upon the thymus is that reported by Jackson in the case of a four-year-old boy, well until six weeks before coming under observation, when there was a sudden croupy attack. This was followed by persistent and increasing stridor and dyspnea. The larynx was free, but a radiograph showed the shadow of an enlarged thymus gland. A tracheotomy was performed and a long canula, reaching almost to the bifurcation was introduced. This gave entire relief, but whenever an attempt was made to remove the tube or to substitute a shorter one, the symptoms recurred. Four weeks after the tracheotomy a second operation was performed and the enlarged thymus was removed with comparative ease. The presence of the long canula in the trachea was a great help in preventing the operative manipulations from interfering with the respirations. The boy made a good recovery and there has been no return of the stridor or dyspnea.

From this review of the reported cases, it is clear that operative intervention in enlargement of the thymus is a safe procedure, even in very young infants. Of the eight cases operated upon only one died, and in that case the inference seems permissible that if the parents had consented to an earlier operation a favorable result might have been obtained.

In July, 1907, Friedlander reported a case of "Status Lymphaticus and Enlargement of the Thymus" occurring in a male infant, in whom whistling inspiration and paroxysms of dyspnea

with cyanosis began to manifest themselves at the age of five weeks. The physical signs of enlarged thymus were very definite. As the symptoms grew steadily worse in spite of inunctions and internal medication, treatment by x-ray exposures was instituted when the child was eight weeks of age. Within two weeks distinct improvement was apparent, and under a continuation of the treatment the progress to complete recovery was continuous and uneventful.

Encouraged by this result, it was thought best to follow a similar course in the case under consideration before suggesting operation. X-ray exposures were begun on September 14, using a tube of moderate hardness, energized by a static machine. The first exposures were made at a distance of twelve inches, but later this was reduced to ten inches. The first exposures were short, only one minute anteriorly and an equal time posteriorly. This was gradually increased as time went on, and during the latter part of the course the front of the chest was given ten minutes and the back five minutes. Six exposures were given in the first eleven days, and as early as this some improvement manifested itself by longer periods of quiet sleep at night and less labored respiration during the day. A rest of ten days was then ordered to await developments, but at the end of five days the mother brought the baby for further x-ray treatment, saying that the trouble had increased in severity as soon as these were discontinued. From this time they were given as nearly every day as the weather permitted. In all, forty-seven exposures were given. Nothing more than a transient erythema at the site of exposure was seen on the skin at any time.

Improvement took place gradually but steadily. The rest became quieter at night, the crowing diminished in intensity, and the periods of freedom became longer, the bowing forward of the sternum and the indrawing of the suprasternal notch and epigastrium gradually diminished. The size of the thymus as indicated by the physical signs steadily grew less.

In the latter part of October the administration of thyroid gland was begun with a twofold purpose—to prevent any nutritional disturbance which might result from a partial interference with the activity of the thyroid gland, as this could not be entirely protected from the rays; and to make use of its antagonistic effect on the thymus, to which Solis-Cohen has directed attention. The improvement, which was already taking place under the x-ray

exposures, continued uninterruptedly, while the thyroid gland was being given and seemed to be perhaps a little accelerated by it, but no definite opinion as to its beneficial action can be given.

The last x-ray exposure was given December 11. By this time the area of thymic dullness was reduced to a half-inch equilateral triangle behind the manubrium. The dull area posteriorly had disappeared long before. The baby slept quietly all night and breathed quietly most of the day, but still had a trace of the inspiratory crow when crying hard or when greatly excited.

Improvement continued after the x-ray treatments were discontinued, and on February 11, 1908, the following note was made: "Normal baby, no thymic dullness, no bowing of sternum with inspiration, no abnormal sound with inspiration, except an occasional little snort."

In conclusion, the interesting features may be summarized as follows: (1) Coexistence of the clinical features of congenital laryngeal stridor, with physical signs strongly suggesting enlargement of the thymus gland; (2) Disappearance of these physical signs under x-ray treatment; (3) Coincident clearing up of the symptoms of laryngeal stridor.

It is not maintained that every case of congenital laryngeal stridor has an enlarged thymus as a causative factor, but it is maintained that such an enlargement may give rise to this symptom complex, and that cases of this character are within the reach of our therapeutic resources.

DUODENAL ENZYMES IN INFANTS WITH KWASHIORKOR OR WITH DYSTROPHIC CONDITION. G. Russo and P. Buttitta. (Minerva pediat., 9:382-392, April 7, 1957). The authors report on the values of the duodenal enzymes in 3 groups of infants. The first group was composed of 8 children with domestic Kwashiorkor. The second group was composed of 12 infants with dystrophy; of these, 11 had hepatic steatosis, 5 riboflavin deficiency, 8 hypodystrotinemia, and 5 edema. The third group was composed of 12 normal children used as controls. The enzyme values were low in the duodenal juice of all the children with Kwashiorkor and in some of the children with dystrophy. The values were higher or became normal after the children had recovered from the disease. The authors believe that the pancreatic alteration that accompanies the deficiency of essential nutritive factors does not cause hepatic steatosis.—*J.A.M.A.*

DEPARTMENT OF ABSTRACTS

Conducted by

MICHAEL A. BRESCIA, M.D., NEW YORK

WYSHAM, D. N.; MULHERN, M. E.; NAVARRE, G. C.; LA VECK, G. D.; KENNAN, A. L. AND GIEDT, W. R.: STAPHYLOCOCCAL INFECTIONS IN AN OBSTETRIC UNIT. I.—EPIDEMIOLOGIC STUDIES OF PYODERMA NEONATORUM. (New England Journal Medicine, 257:296, August 15, 1957).

An epidemic of staphylococcal infections in a nursery was studied in which fifty-four per cent of one hundred and seventeen newborn infants contracted clinical staphylococcal infections. To determine the source of the staphylococci causing this epidemic, daily cultures were taken from the infants in the nursery and from their mothers. Cultures were made frequently from nursery personnel and from fomites and the air in the nursery. The strains of staphylococci isolated were identified by both their phage types and their sensitivity to antibiotics. The majority of infections were caused by a single strain of Staphylococcus aureus, termed the epidemic strain. This strain was of phage type 52/42B/81, and resistant to penicillin, streptomycin and tetracycline. Forty-nine of the infants were colonized by this strain while in the nursery. It was evident that the mothers were not the source of the epidemic for none of them were admitted to the hospital carrying the epidemic strain. Two nursery personnel were carriers of this strain, but both had insufficient contact with infants to have produced the epidemic. Infants who were subclinically infected with Staphylococcus aureus disseminated large numbers of staphylococci into the nursery environment, including the nursery air. Such infants seemed to be the major source of staphylococcal infection. In the spread of infection from infant to infant, aerial transmission appeared to play an important part. Changes in nursery design or the practice of rooming-in for infants appears indicated to effect permanent control.

AUTHOR'S SUMMARY.

GEYER, M. L. AND YANKAUER, A.: DETECTION OF HEARING LOSS IN PRESCHOOL CHILDREN. (Public Health Reports, 72:723, August 1957).

In a study in Rochester, New York, an individual sweep check for hearing loss was successfully administered to all but 6.5 per cent of 461 children from 2½ years through 5½ years of age. Fifty of the fifty-three children who failed the screening test were screened a second time. Twenty-eight of these children failed the second screening test and a subsequent pure tone threshold test. Thus 6.5 per cent of 431 children successfully screened were considered to have a hearing loss. Medical or educational recommendations were made for twenty-one of them. The average time required to condition and screen a child in this study was five and one-half minutes. From the standpoint of prevention, amelioration, and educational therapy, mass screening of the preschool population is worthwhile. The method described here appears to be one way of effectively and quickly screening this population.

AUTHOR'S SUMMARY.

YING-K'UN, F. AND MING-HSÜN, T'AN: THE STURGE-KALISCHER-WEBER SYNDROME. A REPORT OF FOUR CASES. (Chinese Medical Journal, 75:344, May 1957).

Four cases of Sturge-Kalischer-Weber syndrome are reported. All of them had extensive facial, cutaneous, port-wine colored nevi and convulsive seizures. One patient also had hemihypertrophy of the right limbs contralateral to the facial nevus. Another patient was a mental defective, whose parents were first cousins, with a family history of mental deficiency, epilepsy and pigmented nevus. This patient had early optic atrophy increase in the number and anastomoses of retinal veins of the left eye, hypotrophy of the left limbs, inconstant Babinski, Chaddock and Oppenheim signs on the left side, intracranial calcification, and sacralization of the lumbar vertebra. Another patient was delivered prematurely. He had a supernumerary digit of the right hand, was poorly nourished and underdeveloped, and appeared dull and indifferent. He could not speak. The fourth patient presented a history of two syncopal attacks, mental deficiency, marked thickening of the base of the left anterior fossa, failure of development of the left frontal sinus, and a mildly dysrhythmic electroencephalogram. He became psychotic. M.A.B.

PAQUET, E. AND DELAGE, J.: RHEUMATIC MANIFESTATIONS IN HODGKIN'S DISEASE, LEUKEMIA AND ALLIED CONDITIONS. (Canadian Medical Association Journal, 76:927, June 1, 1957).

Osteo-articular pain is a frequent accompaniment of Hodgkin's disease, acute leukemia and multiple myeloma. Particular attention is paid to dorsal, lumbosacral and sacroiliac pain. Clinical, radiological and pathological considerations are presented. No constant relationship exists between painful manifestations and x-ray patterns. The spine is most commonly involved in Hodgkin's disease, and particularly in the lumbar segment, giving rise to low back pain with or without radiation to the legs. Symptoms referrable to the bones and joints are more frequently seen in acute leukemia of children than in the other types of leukemia. Diagnostic difficulty may exist with rheumatic fever. Although infrequent in chronic leukemia, rheumatic pain is more often observed in myelocytic than in lymphocytic types. General data on the number of cases studied and the incidence of rheumatic complaints in these areas are presented. It is concluded that pain is particularly prominent in multiple myeloma and Hodgkin's disease, and that lumbar or dorsolumbar segments of the spine are the most commonly affected. Pain occurs frequently at the onset of acute leukemia and multiple myeloma. AUTHORS' SUMMARY.

RICCI, G.; LO SARDO, S. and CASINI, E.: Treatment of Tetanus with Chlorpromazine. (Aggiornamenti sulle Malattie Da Infezione, 3:191, May-June 1957).

The authors report on fifty-six cases of tetanus, forty cases between the ages of 7 years and 70 years were treated in 1954 and 1955, and sixteen cases between the ages of 3 years and 63 years were treated in 1956. Of the forty treated earlier, twenty-three recovered and seventeen died, and of the sixteen treated later, twelve recovered and four died. The treatment consisted of specific serum, antibiotics and tubocurarine. However, the authors found that the use of chlorpromazine controlled the tetanic contractures very effectively. For best results they began early in the disease with minimal doses of chlorpromazine and increased the dose as needed depending on the response. M.A.B.

BOOK REVIEWS

Conducted by

MICHAEL A. BRESCIA, M.D., NEW YORK

MANAGEMENT OF THE HANDICAPPED CHILD. Edited by Harold Michal-Smith, Ph.D. Cloth. Pp. 276. Price $6.50. New York: Grune and Stratton, 1957.

In this volume, the editor, Harold Michal-Smith, Chief Clinical Psychologist, Flower and Fifth Avenue Hospitals, New York, and Assistant Professor of Pediatrics, New York Medical College, makes claim to not merely a continuation and extension, but also a retitling of his earlier edition. The former, "Pediatric Problems in Clinical Practice" (Grune and Stratton 1954), was primarily intended to interest pediatricians, whereas, the present edition, although broadened in medical scope, has virtually a text book flavor in its interdisciplinary approach. Its former special appeal to the medical profession has expanded to encompass the fields of psychology, social work, speech correction and education. Together, the two editions may be said to cover the field of pediatric problems and their management, and to have attempted to show that, as in the words of Dr. Arnold Gesell, who has written the foreword to this volume, "Adequate management of child handicap begins with early diagnosis followed by periodic supervision at progressive stages of development." Parents who have looked for a book of this nature in their never-ending search for the answers to questions of management of the handicapped child, will find in this volume, a realistic, sympathetic and practical approach to their problems. There is an excellent bibliography of representative books in the field of their interest to be found appended to this edition. An imposing and noteworthy array of contributors and interest-arousing chapter titles tempt the reader to serious reading. In all, there are thirteen chapters, but space does not allow for extensive review of individual chapters. Suffice it to say, the chapters deal in one way or another with etiology, detection, incidence, types of disorder, management and habilitation.

This volume would undoubtedly be incomplete without the inclusion of the well-developed chapter on "Nervous Habits", con-

tributed by its able editor, Dr. Harold Michal-Smith. His theme is oriented toward the pediatrician, in particular, and it urges early recognition of tension-provoking factors in the child-parent relationship most likely to lead to the formation of the so-called "nervous habits" of his patients, which so often cause serious concern to the parent. He also alerts them to symptoms of disorder in the functioning of the total personality and to early detection of the disequilibrium between maturational and accultural factors manifesting itself in one or another of the tension-discharging neurotic habits. The latter, related to oral, motor, or genital areas, may take the form of thumb-sucking, tics, or masturbation, and should not be confused with more serious behavior disorders, which are covered in a previous chapter of the book.

E. ROTHENBERG, M.A.

COMPARATIVE ASPECTS OF HAEMOLYTIC DISEASE OF THE NEWBORN. By G. Fulton Roberts, M.A., M.D. Cloth. Pp. 199. Price $3.00. London: William Heinemann Medical Books, Ltd.; New York: Grune & Stratton, Inc., 1957.

Although this small volume has quite a formidable title, it, nonetheless, is most interesting reading. The historical aspects of the disease, which is the opening chapter of the book, demonstrates the gradual increase in knowledge in any syndrome and how step by step knowledge increases with the final culmination of the correct interpretation of the disease. This probably can be said of practically all knowledge. It was Newton who said he discovered what he did because he stood on the shoulders of giants. Haemolytic disease of the newborn is admittedly a complicated disease, but the author spares us the many intricate blood groups, types and subtypes explaining the disease without becoming lost in symbols. Half the book is devoted to the disease in man dwelling upon, mainly, the anemia, the kernicterus and the serological considerations. The remainder of the book is devoted to the comparative aspects of the disease as seen in the horse, pig and the experimental production of the disease in the guinea-pig, rat, dog and rabbit. The varying aspects of the disease as seen in the different animals and compared to the disease process in man, helps to give a better understanding of its pathogenesis. The book is highly recommended to those who wish to acquire a fuller knowledge of disease.

M.A.B.

PEDIATRIC ROENTGENOLOGY. By M. A. Lassrich, M.D., Prof. R. Prévôt and K. H. Schäfer, M.D. Edited by Dr. K. H. Schäfer. Translated from the German by James T. Case, M.D., D.M.R.E. Cloth. Pp. 333. Illustrated. Price $28.00. New York: Grune & Stratton, Inc., 1957.

This is an excellent book on roentgenology as it pertains to pediatric problems. It is the type of book that both pediatrician and roentgenologist like to have available to supplement other texts that they might have in the same field. The film reproductions are well done and selected for their instructability. The schematic drawings accompanying some of the roentgen films enhance the value of the book. The book is divided into three main parts, one dealing with the thoracic organs, one with the abdominal organs and the third dealing with the skeletal system. No book is expected to cover the entire field of pediatric problems and, therefore, the films dealing with the abdominal problems seemed incomplete, but this does not detract from the usefulness of the text. There is very little text, the authors confining themselves, and wisely so, to a concise caption with each film. The translation is well done.

M.A.B.

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